




# Facial expressions recognition and discrimination in Parkinson's disease

Giulia Mattavelli<sup>1\*</sup> , Edoardo Barvas<sup>2</sup>, Chiara Longo<sup>2</sup>,  
Francesca Zappini<sup>2</sup>, Donatella Ottaviani<sup>3</sup>, Maria Chiara Malaguti<sup>4</sup>,  
Maria Pellegrini<sup>4</sup> and Costanza Papagno<sup>2,5</sup>

<sup>1</sup>NETS, Scuola universitaria superiore IUSS, Pavia, Italy

<sup>2</sup>CeRiN, Centro di Riabilitazione Neurocognitiva, CIMeC, Università di Trento, Rovereto, Italy

<sup>3</sup>Unità Operativa di Neurologia, Ospedale Santa Maria del Carmine, Azienda Provinciale per i Servizi Sanitari, Rovereto, Italy

<sup>4</sup>Dipartimento di Scienze Neurologiche, Ospedale Santa Chiara, Trento, Italy

<sup>5</sup>Dipartimento di Psicologia, Università degli studi di Milano-Bicocca, Milano, Italy

Emotion processing impairment is a common non-motor symptom in Parkinson's Disease (PD). Previous literature reported conflicting results concerning, in particular, the performance for different emotions, the relation with cognitive and neuropsychiatric symptoms and the affected stage of processing. This study aims at assessing emotion recognition and discrimination in PD. Recognition of six facial expressions was studied in order to clarify its relationship with motor, cognitive and neuropsychiatric symptoms. Sensitivity in discriminating happy and fearful faces was investigated to address controversial findings on impairment in early stages of emotion processing. To do so, seventy PD patients were tested with the Ekman 60 Faces test and compared with 46 neurologically unimpaired participants. Patients' performances were correlated with clinical scales and neuropsychological tests. A subsample of 25 PD patients and 25 control participants were also tested with a backward masking paradigm for sensitivity in happiness and fear discrimination. Results showed that PD patients were impaired in facial emotion recognition, especially for fearful expressions. The performance correlated with perceptual, executive and general cognitive abilities, but facial expression recognition deficits were present even in cognitively unimpaired patients. In contrast, patients' sensitivity in backward masking tasks was not reduced as compared to controls. Taken together our data demonstrate that facial emotion recognition, and fear expression in particular, is critically affected by neurodegeneration in PD and related to cognitive abilities; however, it appears before other cognitive impairments. Preserved performances in discriminating shortly presented facial expressions, suggest unimpaired early stages of emotion processing.

Parkinson's disease (PD) is a neurodegenerative disorder featured by the progressive loss of dopaminergic neurons in the substantia nigra (Dauer & Przedborski, 2003). Motor dysfunctions are the characteristic symptoms, but cognitive, behavioural and emotional

\*Correspondence should be addressed to Giulia Mattavelli, Piazza della Vittoria n.15, 27100 Pavia, Italy (email: [giulia.mattavelli@iusspavia.it](mailto:giulia.mattavelli@iusspavia.it)).

dysfunctions are common in patients (Papagno & Trojano, 2018; Trojano & Papagno, 2018). Among the different impairments, emotion processing in PD has been investigated in several studies driven by clinical and theoretical interest (Borg *et al.*, 2012; Heller *et al.*, 2018; Herrera, Cuetos, & Rodríguez-Ferreiro, 2011; Yip, Lee, Ho, Tsang, & Li, 2003). Indeed, emotional impairments are particularly disturbing for patients and their caregivers and have negative impact on quality of life, social and therapeutic interactions (Argaud, Vérin, Sauleau, & Grandjean, 2018; Clark, Nearing, & Cronin-Golomb, 2008; Tickle-Degnen & Lyons, 2004). From a theoretical point of view, PD patients provide specific evidence concerning the role of dopaminergic system and basal ganglia structures in emotion processing (Péron, Dondaine, Le Jeune, Grandjean, & Vérin, 2011). Neuroimaging and neuropsychological researches have described a cortico-subcortical network underpinning facial emotion processing, which encompasses occipitotemporal regions, the cingulate and prefrontal cortices, the amygdala and the basal ganglia (Adolphs, 2002; Mattavelli *et al.*, 2014, 2019). In particular, emotional deficits in PD support the hypothesis that the basal ganglia play a key role in emotion recognition and regulation as part of a cortico-thalamic network crucial for evaluating and monitoring behaviour (Argaud, *et al.*, 2018; Péron *et al.*, 2011). Moreover, the “amygdalar syndrome”, namely the co-occurrence of negative (reduced emotion discrimination and affective reactions) and positive symptoms (hallucinations and anxiety states) associates PD’s symptomatology to dysfunctions of the amygdala and the dopaminergic system (Diederich, Goldman, Stebbins, & Goetz, 2016; Harding, Stimson, Henderson, & Halliday, 2002).

Impaired emotion processing in PD patients has been proved in several studies investigating recognition of facial expressions and emotional prosody (e.g., Ariatti, Benuzzi, & Nichelli, 2008; Kan, Kawamura, Hasegawa, Mochizuki, & Nakamura, 2002; Narme, Bonnet, Dubois, & Chaby, 2011; Ricciardi *et al.*, 2017; Sprengelmeyer *et al.*, 2003). Literature reviews agreed for the presence of deficits in PD patients, but also highlighted inconsistencies concerning, in particular, the selectivity of the deficit for specific emotions (Argaud *et al.*, 2018; Péron *et al.*, 2011). Moreover, controversial results in the previous literature prevent to outline clear hypotheses concerning the relationship of emotion recognition deficits with motor dysfunction, and facial muscle control in particular; similarly, the associations with cognitive deterioration, executive and visuospatial functions or neuropsychiatric symptoms are still unclear (Argaud *et al.*, 2018; Péron *et al.*, 2011). The occurrence of a selective, or at least disproportional, deficit in certain emotions has been suggested by studies reporting reduced performance in recognizing disgust and anger expressions (Sprengelmeyer *et al.*, 2003; Suzuki, Hoshino, Shigemasa, & Kawamura, 2006), in agreement with the involvement of the basal ganglia and dopaminergic mechanisms in processing these two emotions (Adolphs, 2002; Lawrence, Calder, McGowan, & Grasby, 2002). However, meta-analyses showed that all negative emotions (anger, disgust, fear and sadness) are more impaired than positive ones (happiness, surprise) (Coundouris, Adams, Grainger, & Henry, 2019; Gray & Tickle-Degnen, 2010). The relationship between emotion processing and the general severity of the disease is also controversial. A recent review mentioned that reduced performances are more evident in studies with patients at later stages of the disease (Argaud *et al.*, 2018), but meta-analyses showed no significant correlation with motor symptoms, suggesting independent pathophysiological mechanisms in motor and emotional domains (Coundouris *et al.*, 2019; Gray & Tickle-Degnen, 2010). On the other hand, correlations between emotion recognition and facial expression production or facial muscle control support

the hypothesis that reduced simulation and mirror mechanisms affect emotion processing in PD (Marneweck, Palermo, & Hammond, 2014; Ricciardi *et al.*, 2017).

Correlations with cognitive functions are less investigated, and different findings are reported depending on the type of task used for the assessment (Assogna, Pontieri, Caltagirone, & Spalletta, 2008; Gray & Tickle-Degnen, 2010). Lower scores in emotion processing have been related to impaired performances in neuropsychological tests on attention, working memory, executive functions, and general cognition (Assogna *et al.*, 2010; Narme *et al.*, 2011). However, deficits in facial emotion recognition have been reported also in patients performing at the same level as healthy controls in attentional tasks, in patients with no impaired scores at cognitive tests, or after controlling for patients' cognitive abilities (Alonso-Recio, Serrano, & Martín, 2014; Herrera *et al.*, 2011; Pietschnig *et al.*, 2016). Thus, it seems that emotional dysfunctions may occur independently from the general cognitive impairment of patients, although deficits in working memory, attention or executive functions could affect the performances, in particular when emotional tasks load on these abilities (Argaud *et al.*, 2018).

A further debate concerns whether emotion recognition deficits appear in PD as secondary symptoms of mood and anxiety disorders. In particular, depression has a high prevalence in PD patients, it influences social, cognitive and motor symptoms and it is related to emotion processing deficits (Dalili, Penton-Voak, Harmer, & Munafò, 2015; Jankovic, 2008; Sagna, Gallo, & Pontone, 2014). Previous studies reported higher scores in scales on depression in PD patients compared to healthy controls, but results on correlations between depressive scores and emotion recognition are controversial (Clark *et al.*, 2008; Kalampokini, *et al.*, 2018; Marneweck *et al.*, 2014; Narme *et al.*, 2013) and a meta-analysis showed no significant moderation of depression on emotion recognition (Gray & Tickle-Degnen, 2010).

Finally, it has to be noted that emotion processing involves multiple stages, which can be measured by different tasks: sensitivity in discriminating between emotions depends on sensory and visuo-spatial processes and it is assessed by forced-choice tasks, whereas recognition further requires to identify the correct emotion label among several alternatives as assessed by identification tasks (Haxby, Hoffman, & Gobbini, 2000). The majority of previous studies evaluated recognition process in PD patients (Argaud *et al.*, 2018). An earlier meta-analysis (Gray & Tickle-Degnen, 2010) reported larger estimated effect size in discrimination than identification tasks, at odds with a most recent meta-analysis showing little lower, albeit comparable, effect size for identification than discrimination tasks (Coundouris *et al.*, 2019). However, only few studies tested discrimination and recognition performances in the same sample of patients, thus it remains unclear which stages of emotion processing are impaired in PD (Alonso-Recio, Martín, Rubio, & Serrano, 2014; Marneweck *et al.*, 2014; Pell & Leonard, 2005). Unimpaired early perceptual stages related to visual and automatic emotion processing, have been suggested by studies reporting preserved affective priming effects in PD patients (Castner *et al.*, 2007; Wagenbreth, Wattenberg, Heinze & Zaehle, 2016) and by an electrophysiological study showing that reduced emotional response of PD patients was not associated to altered early brain activity (Wieser, Mühlberger, Alpers, Macht, Ellgring, & Pauli, 2006). To the best of our knowledge, no previous studies assessed perceptual sensitivity in discriminating different emotions varying the time of stimuli presentation and applying measures of the signal detection theory (Stanislaw & Todorov, 1999), to elucidate whether PD patients show impaired sensitivity or abnormal time-related threshold in perceiving emotional expressions. Signal detection measures allow to investigate performances in discriminating target from non-target stimuli in dichotomous

forced-choice tasks, taking into account participants' response bias, namely the tendency to respond yes or no for target presence (Stanislaw & Todorov, 1999). The backward masking paradigm is a common task used in combination with signal detection measures to evaluate individual threshold for awareness in visual stimuli perception or neural responses to unconsciously processed stimuli (Liddell, Williams, Rathjen, Shevrin, & Gordon, 2004; Pessoa, Japee, & Ungerleider, 2005; Williams *et al.*, 2004). In this type of task, a first visual stimulus, e.g., a target emotional face, is briefly presented and followed by a different stimulus of longer duration, e.g., a neutral face, which interrupts the perceptual processing of the first stimulus and limits its access to conscious report when target duration is below the individual threshold of sensitivity (Pessoa *et al.*, 2005). This paradigm has been used to assess individual variability in thresholds for perceptual awareness of emotional faces (Pessoa *et al.*, 2005) and automatic processing of emotional faces in behavioural and neuroimaging studies (Liddell *et al.*, 2004; Roesch, Sander, Mumenthaler, Kerzel, & Scherer, 2010; Williams *et al.*, 2004). In this study, we used a backward masking paradigm to evaluate in patients with PD and control participants the ability of emotions discrimination related to early perceptual capacity. A better understanding of the impaired stage of processing in PD is relevant from a clinical point of view to define the most appropriate task to measure patients' deficits. Moreover, neural pathways underpinning perceptual processing and recognition of emotions are partially distinct (Tamietto & de Gelder, 2010), thus it is of interest to clarify which stage of processing is impaired in PD to infer the underlying neuroanatomical correlates. In the light of the above, the present study aims at clarifying emotion recognition and sensitivity in emotion discrimination in patients with PD, addressing the issue of non-overlapping impairments in early perceptual processing and recognition of emotions. The ability to identify the six basic emotions was assessed to define emotions recognition deficits and evaluate the relationship with motor, cognitive and neuropsychiatric symptoms. Moreover, backward masking tasks were used to explore sensitivity to happy and fearful facial expressions varying presentation durations.

## Methods

### Participants

The study included 70 right-handed patients with PD (44 males, age:  $M = 67.97$ ,  $SD = 8.2$ , years of education  $M = 10.29$ ,  $SD = 4.79$ ) and 46 right-handed neurologically unimpaired participants (22 males, age:  $M = 65.41$ ,  $SD = 6.33$ , years of education  $M = 11.98$ ,  $SD = 4.31$ ). The two groups did not significantly differ for age,  $t(114) = -1.79$ ,  $p = .08$ , years of education,  $t(114) = 1.94$ ,  $p = .06$ , and males versus females proportion,  $\chi^2(1) = 2.56$ ,  $p = .11$ . PD patients were evaluated with the Montreal Cognitive Assessment (MoCA) for the presence of general cognitive impairments and only patients with adjusted score  $> 10$  were included (Conti, Bonazzi, Laiacona, Masina, & Coralli, 2015). MoCA scores, as scores at other neuropsychological tests, were adjusted for demographic variables, according to normative data available for the Italian population (Conti *et al.*, 2015). In particular, raw scores are adjusted for age, education and, when indicated, for gender, according to the parameters estimated in a normal sample (200–321 neurologically unimpaired subjects) with a multiple regression model. Adjusted scores below 5% one-sided non-parametric tolerance limit (with 95% CI) are considered pathological: inferential cut-off scores are therefore those at which or below which the probability that an individual belongs to the normal population is  $< .05$ .

A subsample of 25 PD patients (16 males, age:  $M = 67.36$ ,  $SD = 6.26$ , years of education  $M = 11.48$ ,  $SD = 5.13$ ) and 25 control participants (12 males, age:  $M = 66.36$ ,  $SD = 5.82$ , years of education  $M = 10.12$ ,  $SD = 4.11$ ) completed the backward masking tasks. This subsample was selected on the basis of patients' willingness to participate to the experimental paradigm in addition to the clinical and neuropsychological assessment. The two groups did not significantly differ for age,  $t(48) = -0.58$ ,  $p = .56$ , years of education,  $t(48) = -1.03$ ,  $p = .31$ , and gender ratio,  $\chi^2(1) = 1.3$ ,  $p = .25$ . All participants provided informed consent for participation, which was obtained according to the Declaration of Helsinki and the study was approved by the local Ethical Committee.

### **Clinical and neuropsychological assessment**

Patients' evaluation included tests on executive, attentional, verbal, visuospatial and visuo-perceptive functions: the Stroop test (Caffarra, Vezzadini, Dieci, Zonato, & Venneri, 2002), Trail Making test (TMT, Giovagnoli *et al.*, 1996), phonological verbal fluency (Carlesimo *et al.*, 1995), line orientation judgment test (Benton, Sivan, Hamsher, & Varney, 1994), the unknown face recognition test (Benton & Van Allen, 1968). Moreover, the following scales for neuropsychiatric symptoms were administered: Geriatric Depression Scale (GDS, Galeoto *et al.*, 2018; Sheikh & Yesavage, 1986), Parkinson Anxiety Scale (PAS, Leentjens *et al.*, 2014), Interpersonal Reactivity Index (IRI, Davis, 1983) and the Questionnaire for Impulsive-Compulsive Disorders in Parkinson's Disease–Rating Scale (QUIP, Weintraub *et al.*, 2012). The battery for patients' assessment was integrated over time with clinical scales and tests; for this reason, or for time restriction in testing patients, data were not complete for the whole sample. Tables 1 and 2 and

**Table 1.** Clinical data of PD patients

	<i>N</i> /70	Mean ( <i>SD</i> )	Range	<i>N</i> > cut off
LEDD	70	722.5 (407)	Na	Na
H&Y	70	2.3 (0.7)	1–5	Na
Years Ons	70	7.2 (5.3)	Na	Na
GDS	49	9.7 (6.5)	0–30	21
PAS Pe	49	8.3 (4.3)	0–20	37
PAS E	49	3.6 (3.1)	0–16	17
PAS A	49	2.7 (2.3)	0–12	18
PAS tot	49	14.6 (7.9)	0–48	37
IRI F	32	12.1 (4.9)	0–28	Na
IRI PT	32	17.5 (4.5)	0–28	Na
IRI EC	32	20.2 (4)	0–28	Na
IRI PD	32	9.7 (5.6)	0–28	Na
QUIP P	30	12.8 (14.2)	0–112	Na
QUIP CG	28	11.0 (12.7)	0–112	Na

Note. A = Avoidance; CG = Caregivers' rating; E = Episodic; EC = Empathic concern; F = Fantasy; GDS = Geriatric Depression Scale; H&Y = Hoehn and Yahr scale; IRI = Interpersonal Reactivity index; LEDD = Levodopa equivalent daily dose (mg); Na = not applicable; P = Patients' self-rating; PAS = Parkinson Anxiety scale; Pe = Persistent; PD = Personal distress; PT = Perspective taking; QUIP = Questionnaire for Impulsive-Compulsive Disorders in Parkinson's Disease; Tot = Total score; Year Ons = years from onset.

Table S1 report patients' clinical data and neuropsychological tests, including number of patients with impaired performances according to normative data. Table S2 reports data for the subsample of 25 patients, which completed the backward masking tasks.

### Facial expression tasks

The Ekman 60 Faces test (Dodich *et al.*, 2014; Young, Perrett, Calder, Sprengelmeyer, & Ekman, 2002) was used to assess emotion recognition. Stimuli were presented one at a time at the centre of the computer screen and participants were asked to select which of the six labels provided below the picture (surprise, happiness, fear, disgust, anger, and sadness) best described the emotional expression. Each face remained on the screen until participants responded by pressing the key (from 1 to 6) corresponding to the selected emotion label.

A backward masking paradigm (Figure 1) was used in a subgroup of participants to evaluate sensitivity in discriminating briefly presented happy and fearful emotions. Target stimuli consisted of five models from the Ekman set (F5, F6, F8, M1 and M6) with happy and fearful expressions; moreover, faces with 25% happy expression produced by morphing procedure along the neutral-happy continuum were used as masks, since they are perceived as neutral without the slightly hostile expression of a pure neutral face (Mattavelli *et al.*, 2014). In each trial a 300 ms fixation cross was presented, followed by the target stimulus for 17, 34, 51, 84, 167 or 334 ms and the mask for 100 ms (Pessoa *et al.*, 2005; Williams *et al.*, 2004); then participants were asked to press the 1 or 2 keyboard buttons to answer whether they saw the target emotion or not, and finally to rate on a 6-point Likert scale their confidence in the answer. In two separate blocks, in counterbalanced order across participants, the target emotion was happiness or fear, i.e., fear and happy faces were presented as stimuli in both blocks, but in one block participants were asked whether or not they saw fearful faces, in the other block they were asked whether or not they saw happy faces. Each block consisted of 240 trials, i.e., 40 trials for each duration of target/non-target presentation in random order.

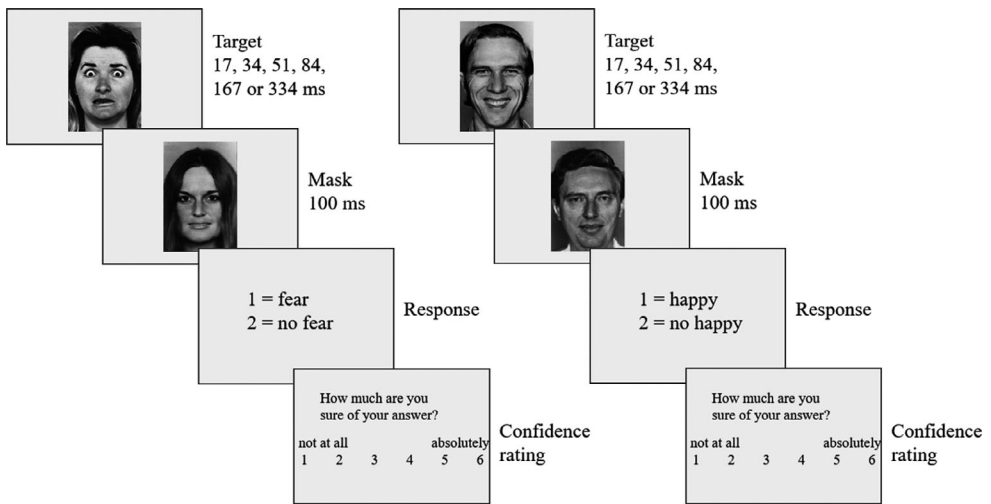
**Table 2.** Neuropsychological data of PD patients

	Cut-off scores	N/70	Mean (SD)	N < cut off
MoCA	<17.36	69	21.2 (3.9)	14
Stroop E	≥4.24	69	3.5 (6.2)	16
Stroop T	≥36.92	69	28.8 (25)	13
TMT A	>93	70	49.2 (51.5)	7
TMT B <sup>a</sup>	>281	58	130.3 (84)	18/70
TMT BA <sup>a</sup>	>186	58	94.6 (76.5)	19/70
Phon Flu	<17.35	70	35.1 (12.8)	1
Line Judg	<19	66	22.6 (5.8)	14
Benton Face	<39	28	45.0 (5.3)	5

Notes. Stroop E = errors; Stroop T = time; Phon Flu = phonological fluency; Line Judg = line orientation judgment test; Benton Face = unknown face recognition.

<sup>a</sup>TMT B and BA were administered to all participants, but when patients could not conclude within 5 min it was interrupted, thus some patients do not have the corrected score, although they were classified as impaired.





**Figure 1.** Timelines with stimuli examples of backward masking tasks when fear expression was the target (left side) and when happy expression was the target (right side).

### Data analysis

Analyses were performed with SPSS statistical software (version 25; IBM Corp, Armonk, NY, USA).

Performances at the Ekman test were analysed by introducing accuracy as dependent variable in the ANOVA Emotion (within-subjects factor, six levels)  $\times$  Group (between-subjects factor, two levels).

In order to evaluate the relationship between emotion recognition and PD symptoms, bootstrap correlation analyses (1,000 resampling with replacement) were carried out between patients' clinical variables, corrected scores at neuropsychological tests and performances at the Ekman 60 faces test. 95% bias-corrected (BCa) confidence intervals, computed for Pearson or Spearman test where appropriate, were considered and the null hypothesis of zero correlation is rejected when confidence intervals do not include 0.

Performances at the backward masking tasks were analysed computing for each target emotion and presentation duration the percentage of accuracy (ACC), false alarms rate (FA) and  $d'$ , which is the index of sensitivity according to the signal detection theory (Stanislaw & Todorov, 1999). These dependent variables were introduced in three ANOVAs Emotion (within-subjects factor, two levels)  $\times$  Duration (within-subjects factor, six levels)  $\times$  Group (between-subjects factor, two levels). Moreover, rating scores on confidence were analysed by dividing ratings when the response was a correct detection (hit-rating) and ratings in FA responses (FA-rating). Hit-rating was analysed by means of an ANOVA Emotion (two levels)  $\times$  Duration (6 levels)  $\times$  Group (two levels). FA-rating was computed for the two emotions averaged across the six durations of presentation and were analysed with an ANOVA Emotion (two levels)  $\times$  Group (two levels). In all ANOVAs Greenhouse–Geisser correction to degrees of freedom was applied when appropriate, *post hoc* analyses were run with Bonferroni correction for multiple comparison and significance threshold was set at .05.

Results

Ekman 60 Faces test

Accuracy for each emotion separately, and the global score adjusted for demographic variables are reported in Table 3. According to normative data for the Italian population (Dodich *et al.*, 2014) four (5.7%) PD patients scored below the cut-off (<37.47) and eight (11.4%) patients had a borderline equivalent score (ES)<sup>1</sup> of 1; whereas only one (2.2%) healthy control scored below the cut-off. Considering cut-off scores separately for each emotion, 24 (34.3%) PD patients were impaired in fear recognition, nine (12.8%) in anger, seven (10%) in happiness and three (4.3%) in surprise, disgust and sadness recognition. In the control group 3 (6.5%) participants scored below the cut-off for fear recognition, two (4.3%) for happiness and one (2.2%) for angry expressions.

The ANOVA (see Figure 2) showed significant effects of Emotion,  $F(3.75, 427.51) = 175.82, p < .001$ , partial  $\eta^2 = .61$ , Group,  $F(1, 114) = 34.33, p < .001$ , partial  $\eta^2 = .23$ , and Emotion  $\times$  Group interaction,  $F(3.75, 427.51) = 6.69, p < .001$ , partial  $\eta^2 = .05$ . PD patients were overall less accurate than healthy controls; *post hoc* tests for the main effect of Emotion showed significant higher accuracy for happiness than all other expressions (all  $ps < .001$ ), whereas the accuracy score for fear was significantly lower than for all other emotions (all  $ps < .001$ ); moreover, surprise was significantly better recognized than disgust, anger and sadness (all  $ps < .001$ ), and anger was significantly less recognized than disgust and sadness ( $ps < .001$ ). *Post hoc* tests for the Emotion by Group interaction revealed that PD patients' accuracy was lower than control group in all emotions (all  $ps < .01$ ) but happiness ( $p = .45$ ).

Relationship between emotion recognition and clinical variables

Bootstrap correlation analyses (Table 4) revealed that the Ekman global score was negatively correlated with the Hoehn and Yahr scale, TMT B, TMT BA and GDS, whereas

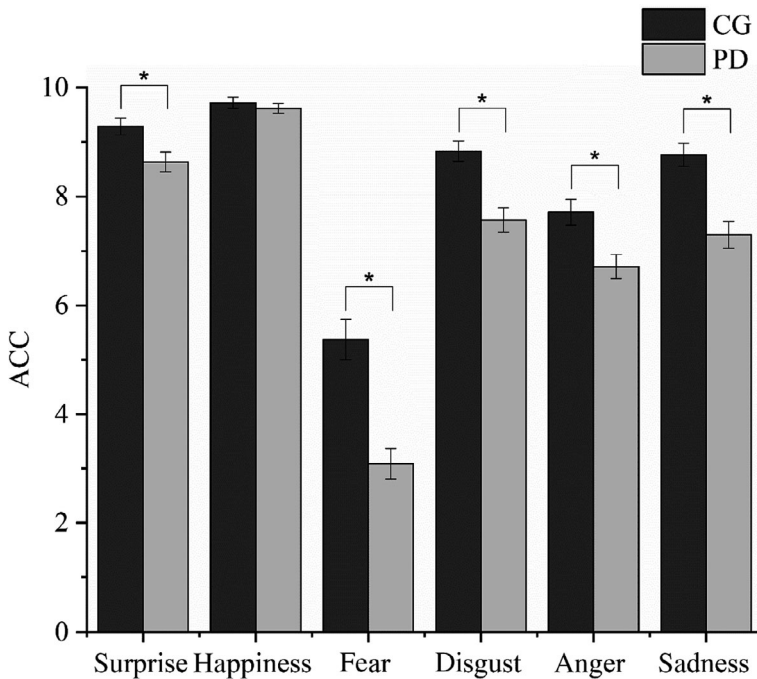
**Table 3.** Performances at the Ekman 60 Faces test expressed as adjusted global score and sub-scores for single emotions. Mean and standard deviation in brackets are reported for patients (PD) and control group (CG)

	PD	CG
Global score	47.41 (5.94)	53.09 (4.87)
Surprise <sup>a</sup>	8.63 (1.52)	9.28 (1.05)
Happiness	9.61 (0.75)	9.72 (0.69)
Fear <sup>a</sup>	3.09 (2.34)	5.37 (2.53)
Disgust <sup>a</sup>	7.57 (1.88)	8.83 (1.27)
Anger <sup>a</sup>	6.71 (1.88)	7.72 (1.64)
Sadness <sup>a</sup>	7.3 (2.06)	8.76 (1.43)

Note. <sup>a</sup>Indicates significant difference in *post hoc* analysis on the Emotion by Group interaction.

<sup>1</sup> Equivalent scores (ES) correspond to a five-point interval scale from 0 to 4 defined on the basis of demographical adjusted scores and non-parametric tolerance limits. An ES of 0 corresponds to performances below the fifth centile of the normal population, whereas an ES of 4 is equal or above the median. An ES of 1 is between the outer and inner tolerance limits and the performance is considered borderline. ES 2 and 3 are intermediate values on a quasi-interval scale and define normal performances (Capitani & Laiacona, 1997).





**Figure 2.** Mean accuracy for control group (CG) and patients (PD) in each emotion of the Ekman 60 Faces test. Bars represent standard error of the means. \* Indicates significant difference in *post hoc* analysis on the Emotion by Group interaction.

positive correlations resulted with the MoCA, verbal fluency on phonological cue, line orientation judgment test, unknown face recognition test and the IRI subscale on empathic concern. Looking at scores for recognition of single emotions, surprise correlated only with the unknown face recognition test; happiness with Stroop errors, verbal fluency on phonological cue and unknown face recognition test; fear with the Hoehn and Yahr scale, MoCA, Stroop errors, verbal fluency and line orientation judgment test; disgust with the MoCA, Stroop errors and time, phonological verbal fluency and line orientation judgment test; anger with the MoCA, Stroop time and unknown face recognition test; sadness with the MoCA, Stroop errors and time, phonological verbal fluency, line orientation judgment test, unknown face recognition test and the IRI subscale on empathic concern. To further investigate the possibility that emotion recognition capacity depends on patients' general cognitive ability we repeated the analysis on the Ekman test, comparing healthy controls and patients, but dividing the patients' sample in subgroups of cognitively unimpaired patients, namely those with MoCA ES > 2 ( $N = 33$ ), and patients with cognitive deterioration, namely MoCA ES < 2 ( $N = 23$ ) (see Note 1 for ES definition). Thus, an ANOVA Emotion (within-subjects factor, six levels)  $\times$  Group (between-subjects factor, three levels) was performed. In cognitively preserved patients, one (3%) patient was impaired in the global score of the Ekman test, 11 (33.3%) patients scored below the cut-off in fear, two (6%) in happiness and anger, one (3%) in surprise and none in disgust and sadness recognition. In the group of patients with MoCA ES < 2, two patients (8.7%) were impaired in the global score, nine (39.1%) patients scored below the cut-off in fear, four (17.4%) in anger, three (13%) in happiness, two

**Table 4.** Bias-corrected 95% confidence intervals (CIs) of bootstrap correlation analysis between scores in emotion recognition and neuropsychological and clinical scales in PD patients

	Ekman GS	Surprise	Happiness	Fear	Disgust	Anger	Sadness
LED D	-.21 .18	-.21 .2	-.13 .3	-.19 .26	-.15 .29	-.19 .22	-.18 .19
H&Y <sup>a</sup>	-.50 <b>-.06</b>	-.32 .14	-.39 .05	<b>-.59</b> <b>-.22</b>	-.44 .07	-.43 .01	-.39 .04
Years Onset	-.28 .19	-.18 .22	-.12 .28	-.09 .38	-.16 .24	-.45 .09	-.10 .28
MoCA	<b>.19 .53</b>	-.11 .33	-.1 .29	<b>.03 .51</b>	<b>.01 .46</b>	<b>.03 .45</b>	<b>.16 .51</b>
Stroop E	-.45 .06	-.38 .1	<b>-.66</b> <b>-.03</b>	<b>-.34</b> <b>-.05</b>	<b>-.39</b> <b>-.06</b>	-.45 .01	<b>-.49</b> <b>-.09</b>
Stroop T	-.39 .05	-.24 .21	-.48 .17	-.24 .21	-.44 <b>-.1</b>	<b>-.43</b> <b>-.02</b>	<b>-.42</b> <b>-.11</b>
TMT A	-.15 .22	-.22 .16	-.38 .08	-.26 .32	-.26 .09	-.15 .17	-.39 .15
TMT B	<b>-.46</b> <b>-.07</b>	-.42 .2	-.19 .2	-.37 .01	-.24 .18	-.43 .002	-.5 .04
TMT BA	<b>-.45</b> <b>-.09</b>	-.4 .19	-.14 .23	-.37 .001	-.25 .15	-.43 .02	-.52 .05
Phon Flu	<b>.17 .55</b>	-.08 .34	<b>.1 .37</b>	<b>.15 .58</b>	<b>.09 .43</b>	-.004 .41	<b>.05 .46</b>
Line Judg	<b>.17 .54</b>	-.01 .48	-.16 .4	<b>.1 .51</b>	<b>.27 .56</b>	-.02 .48	<b>.04 .53</b>
Benton Face	<b>.24 .79</b>	<b>.16 .7</b>	<b>.06 .69</b>	-.11 .57	-.15 .56	<b>.15 .73</b>	<b>.21 .71</b>
GDS	<b>-.46</b> <b>-.01</b>	-.25 .25	-.22 .17	-.38 .08	-.3 .17	-.41 .15	-.37 .15
PAS Pe	-.3 .35	-.2 .4	-.26 .27	-.16 .43	-.3 .32	-.39 .23	-.42 .16
PAS E	-.45 .07	-.41 .14	-.24 .32	-.24 .38	-.4 .11	-.35 .12	-.54 .09
PAS A	<b>-.36 .08</b>	-.38 .08	-.14 .37	-.35 .18	-.19 .33	-.28 .22	-.54 .1
PAS Tot	-.39 .16	-.34 .26	-.26 .32	-.25 .35	-.33 .22	-.37 .16	-.24 .14
IRI F	-.14 .53	-.22 .5	-.15 .37	-.52 .48	-.14 .55	-.16 .4	-.27 .29
IRI PT	-.34 .35	-.58 .05	-.26 .34	-.15 .54	-.11 .42	-.19 .49	-.31 .38

*Continued*

**Table 4.** (Continued)

	Ekman GS	Surprise	Happiness	Fear	Disgust	Anger	Sadness
IRI EC	<b>.18 .69</b>	-.18 .62	-.05.34	-.04 .59	-.02 .41	-.1 .58	<b>.09 .57</b>
IRI PD	-.49 .23	-.37 .23	-.44 .51	-.45 .14	-.31 .33	-.54 .28	-.49 .03
QUIP P	-.17 .32	-.18 .52	-.61 .3	-.37 .31	-.25 .53	-.13 .52	-.07 .47
QUIP CG	-.11 .56	-.18 .47	-.38 .23	-.01 .59	-.15 .32	-.31 .51	-.35 .38

Notes. A = Avoidance; Benton Face = unknown face recognition; CG = Caregivers' rating; E = Episodic; EC = Empathic concern; Ekman GS = global score; F = Fantasy; GDS = Geriatric Depression Scale; H&Y = Hoehn and Yahr scale; IRI = Interpersonal Reactivity index; LEDD = Levodopa equivalent daily dose; Line Judg = line orientation judgment test; P = Patients' self-rating; PAS = Parkinson Anxiety scale; Pe = Persistent; PD = Personal distress; Phon Flu = phonological fluency; PT = Perspective taking; QUIP = Questionnaire for Impulsive-Compulsive Disorders in Parkinson's Disease; Stroop E = errors; Stroop T = time; Tot = Total score; Years Onset = years from onset.

The two values per cell are the lower and upper CIs. The null hypothesis of zero correlation is rejected when confidence intervals do not include 0, thus variables showing unidirectional negative or positive correlations are highlighted in bold

Table <sup>a</sup> CIs for the Hoehn and Yahr scale were computed for Spearman's rank order correlation.

(8.7%) in disgust and sadness and one (4.3%) in surprise recognition. As above, the effects of Emotion,  $F(3.5, 353.7) = 173.08, p < .001$ , partial  $\eta^2 = .64$ , Group,  $F(2, 99) = 25.05, p < .001$ , partial  $\eta^2 = .34$ , and their interaction,  $F(7.1, 353.7) = 4.1, p < .001$ , partial  $\eta^2 = .08$ , were significant. *Post hoc* tests for the Group main effect showed that all comparisons between the three groups were significant, with the control group performing better than cognitively preserved ( $p = .001$ ) and cognitively impaired patients ( $p < .001$ ); moreover, cognitively preserved patients performed better than cognitively impaired patients ( $p = .002$ ). *Post hoc* analysis on Emotion by Group interaction revealed that cognitive impaired PD patients had a significantly lower accuracy than the control group for all emotions ( $ps < .01$ ), but happiness ( $p = .1$ ). In fear recognition, also cognitively unimpaired patients had lower accuracy than the control group ( $p = .005$ ) and did not differ from cognitively impaired PD group ( $p = .15$ ), whereas only in sadness recognition cognitively unimpaired patients performed significantly better than cognitively impaired PD group ( $p = .002$ ).

### **Backward masking paradigm**

Detailed results and statistics are reported in Tables 5 and 6. The main effects of Emotion and Duration were significant in the analysis on ACC, which was higher for happiness ( $M = 0.64, SD = 0.27$ ) than fear ( $M = 0.51, SD = 0.28$ ) discrimination and was lower for 17 and 34 ms presentation compared to all other presentation durations ( $ps < .001$ ); ACC significantly increased between 51, 84, 167 and 334 ms presentation (all  $ps < .001$ ). The factor Group was not significant as main effect or in interaction with other factors.

FA analysis showed significant main effects of Emotion and Duration, being FA rate lower for fear ( $M = 0.19, SD = 0.15$ ) than happiness ( $M = 0.26, SD = 0.2$ ) recognition, and lower for 334 ms and 167 ms compared to faster presentations ( $ps < .001$ ). Moreover, *post hoc* tests for the significant Emotion  $\times$  Duration interaction revealed that FA rate was higher for happy than fear emotion at 17 ms ( $p = .007$ ), 34 ms ( $p = .002$ ) and 51 ms ( $p = .009$ ) presentation, but not at longer durations ( $ps > .05$ ). The effect of Group was not significant.

Similarly to FA, analysis on  $d'$  showed significant effects of Emotion, Duration and Emotion  $\times$  Duration interaction, whereas Group was not significant as main effect or in interaction with other factors.  $d'$  was higher for happiness ( $M = 1.33, SD = 1.29$ ) than fear ( $M = 1.07, SD = 1.15$ ) discrimination. *Post hoc* tests showed significantly lower  $d'$  for 17 and 34 ms presentation compared to all other presentation durations ( $ps < .001$ ), then  $d'$  significantly increased between 51, 84, 167 and 334 ms presentation (all  $ps < .001$ ). Moreover,  $d'$  was significantly higher for happy than fear target emotion at 167 ms and 334 ms presentation ( $ps < .001$ ), but not at shorter presentation durations ( $ps > .05$ ) (Figure 3).

Responses at rating on confidence following hit or false alarm trials were also analysed. Since some of the participants did not produce hit responses in 17 ms or 34 ms duration conditions, hit-rating analyses included 21 PD patients and 21 controls. The main effects were significant: Hit-rating were higher following the detection of happy ( $M = 4.93, SD = 1.11$ ) than fear ( $M = 4.62, SD = 1.19$ ) expression and were higher in the control ( $M = 5.11, SD = 1.07$ ) than in PD group ( $M = 4.44, SD = 1.16$ ). *Post hoc* tests for the main effect of Duration showed that Hit-rating was higher for 334 and 167 ms conditions compared to all shorter durations ( $ps < .001$ ). In the analysis on FA-rating the main effect of Emotion was significant, whereas the main effect of Group or Group  $\times$  Emotion interaction were not significant. FA-rating was higher following FA responses in blocks

**Table 5.** TablePerformances at backward masking tasks for fear and happiness discrimination. Mean and standard deviation in brackets are reported for patients (PD) and control group (CG)

Duration	ACC		FA		d'		Hit-rating	
	PD	CG	PD	CG	PD	CG	PD	CG
Fear								
17 ms	0.26 (0.19)	0.28 (0.19)	0.26 (0.16)	0.20 (0.15)	-0.02 (0.51)	0.21 (0.34)	4.2 (1.24)	4.79 (1.25)
34 ms	0.26 (0.16)	0.31 (0.2)	0.25 (0.15)	0.23 (0.2)	0.04 (0.46)	0.31 (0.69)	3.88 (1.36)	4.62 (1.29)
51 ms	0.38 (0.18)	0.38 (0.19)	0.22 (0.15)	0.24 (0.17)	0.56 (0.55)	0.47 (0.51)	4.01 (1.26)	4.74 (1.14)
84 ms	0.56 (0.19)	0.55 (0.2)	0.23 (0.14)	0.20 (0.17)	1.03 (0.79)	1.14 (0.82)	4.04 (1.23)	4.94 (0.87)
167 ms	0.74 (0.21)	0.77 (0.15)	0.15 (0.1)	0.15 (0.12)	1.92 (0.85)	2.01 (0.73)	4.53 (1.04)	5.32 (0.77)
334 ms	0.83 (0.15)	0.85 (0.13)	0.10 (0.09)	0.09 (0.08)	2.51 (0.88)	2.69 (0.75)	4.91 (0.92)	5.42 (0.74)
Happiness								
17 ms	0.39 (0.22)	0.37 (0.21)	0.34 (0.2)	0.31 (0.19)	0.15 (0.34)	0.21 (0.36)	4.36 (1.03)	5.01 (1.32)
34 ms	0.47 (0.2)	0.44 (0.19)	0.33 (0.2)	0.34 (0.19)	0.40 (0.52)	0.27 (0.36)	4.21 (1.03)	4.95 (1.16)
51 ms	0.52 (0.2)	0.54 (0.18)	0.32 (0.21)	0.33 (0.16)	0.73 (0.82)	0.61 (0.5)	4.34 (1.07)	5.03 (1.21)
84 ms	0.61 (0.24)	0.73 (0.17)	0.26 (0.2)	0.30 (0.19)	1.14 (0.83)	1.36 (0.86)	4.46 (1.1)	5.2 (1.02)
167 ms	0.85 (0.18)	0.89 (0.14)	0.17 (0.15)	0.15 (0.14)	2.35 (0.94)	2.59 (0.83)	5.02 (0.91)	5.54 (0.78)
334 ms	0.93 (0.1)	0.94 (0.11)	0.11 (0.13)	0.10 (0.13)	3.04 (0.77)	3.12 (0.73)	5.3 (0.96)	5.7 (0.59)

Note. ACC = accuracy; FA = false alarm.

**Table 6.** Results of statistical analysis of the backward masking tasks

Effect	Statistic
Accuracy	
Emotion	$F(1, 48) = 35.45, p < .001, \text{partial } \eta^2 = .42^*$
Duration	$F(2.62, 125.83) = 214.88, p < .001, \text{partial } \eta^2 = .82^*$
Group	$F(1, 48) = 0.6, p = .44, \text{partial } \eta^2 = .01$
Emotion $\times$ Group	$F(1, 48) = 0.025, p = .87, \text{partial } \eta^2 < .01$
Duration $\times$ Group	$F(2.62, 125.83) = 0.37, p = .75, \text{partial } \eta^2 = .01$
Emotion $\times$ Duration	$F(2.43, 116.61) = 1.27, p = .29, \text{partial } \eta^2 = .03$
Emotion $\times$ Duration $\times$ Group	$F(2.43, 116.61) = 1.83, p = .15, \text{partial } \eta^2 = .04$
False alarm	
Emotion	$F(1, 48) = 6.08, p = .017, \text{partial } \eta^2 = .11^*$
Duration	$F(2.86, 137.33) = 49.67, p < .001, \text{partial } \eta^2 = .51^*$
Group	$F(1, 48) = 0.9, p = .77, \text{partial } \eta^2 < .01$
Emotion $\times$ Group	$F(1, 48) = 0.17, p = .68, \text{partial } \eta^2 < .01$
Duration $\times$ Group	$F(2.86, 137.33) = 0.88, p = .45, \text{partial } \eta^2 = .02$
Emotion $\times$ Duration	$F(3.99, 191.37) = 4.5, p = .002, \text{partial } \eta^2 = .09^*$
Emotion $\times$ Duration $\times$ Group	$F(3.99, 191.37) = 0.57, p = .68, \text{partial } \eta^2 = .01$
D'	
Emotion	$F(1, 48) = 16.09, p < .001, \text{partial } \eta^2 = .25^*$
Duration	$F(3.48, 167.18) = 310.06, p < .001, \text{partial } \eta^2 = .87^*$
Group	$F(1, 48) = 0.77, p = .38, \text{partial } \eta^2 = .02$
Emotion $\times$ Group	$F(1, 48) = 0.36, p = .55, \text{partial } \eta^2 < .01$
Duration $\times$ Group	$F(3.48, 167.18) = 0.67, p = .59, \text{partial } \eta^2 = .01$
Emotion $\times$ Duration	$F(5, 240) = 3.2, p = .008, \text{partial } \eta^2 = .06^*$
Emotion $\times$ Duration $\times$ Group	$F(5, 240) = 0.97, p = .44, \text{partial } \eta^2 = .02$
Hit-rating	
Emotion	$F(1, 40) = 6.65, p = .014, \text{partial } \eta^2 = .14^*$
Duration	$F(2.59, 103.57) = 26.98, p < .001, \text{partial } \eta^2 = .40^*$
Group	$F(1, 40) = 6.15, p = .017, \text{partial } \eta^2 = .13^*$
Emotion $\times$ Group	$F(1, 40) = 0.14, p = .71, \text{partial } \eta^2 < .01$
Duration $\times$ Group	$F(2.59, 103.57) = 0.82, p = .47, \text{partial } \eta^2 = .02$
Emotion $\times$ Duration	$F(3.06, 122.37) = 0.45, p = .72, \text{partial } \eta^2 = .01$
Emotion $\times$ Duration $\times$ Group	$F(3.06, 122.37) = 0.43, p = .73, \text{partial } \eta^2 = .01$
False alarm-rating	
Emotion	$F(1, 48) = 6.22, p = .016, \text{partial } \eta^2 = .11^*$
Group	$F(1, 48) = 0.08, p = .77, \text{partial } \eta^2 < .01$
Emotion $\times$ Group	$F(1, 48) = 0.33, p = .57, \text{partial } \eta^2 < .01$

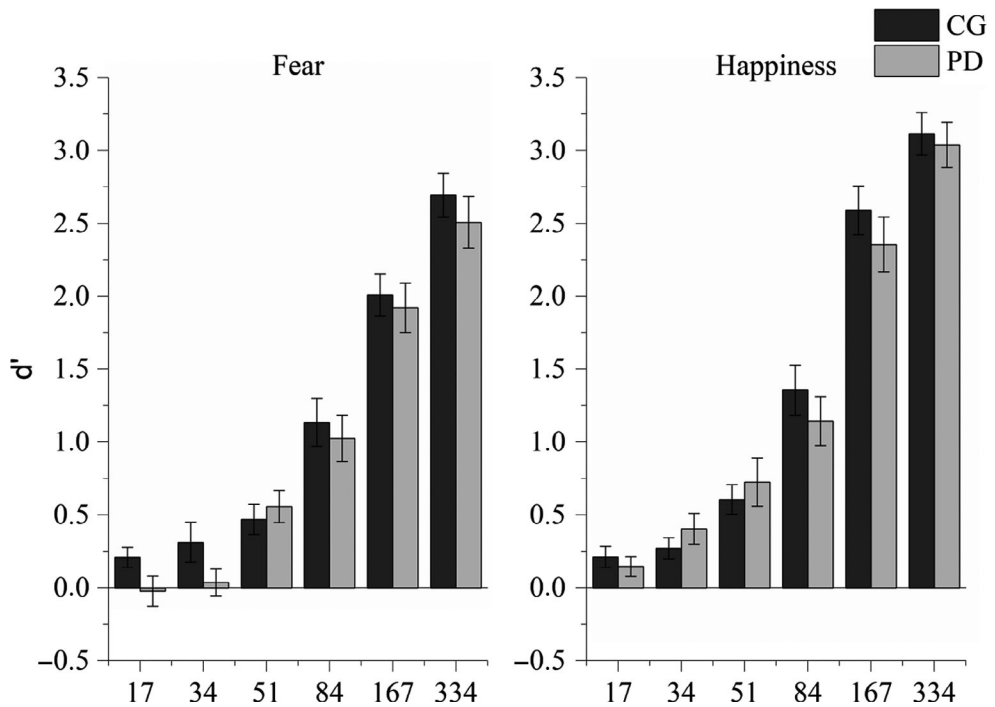
Note.  $*p < .05$ .

where happy expression was the target ( $M = 4.43, SD = 1.13$ ) compared with blocks with fearful expression as target ( $M = 4.03, SD = 1.36$ ).

## Discussion

This study examined emotion recognition and discrimination in PD patients. The main findings can be summarized as follows: (1) PD patients scored worse than healthy controls in recognizing all basic emotions, but happiness; (2) performances in emotion





**Figure 3.** Mean  $d'$  for control group (CG) and patients (PD) in each duration of presentation (ms) in the backward masking tasks on fear and happiness discrimination. Bars represents standard error of the means.

discrimination using a backward masking paradigm were not significantly different between patients and healthy controls; (3) global score of PD patients in emotion recognition correlated with the stage of the disease, and scores at the MoCA, TMT, verbal fluency on phonological cue, line orientation judgment, unknown face recognition tests and the empathic concern subscale of the IRI; (4) a weak correlation resulted with depression whereas clinical scales on other neuropsychiatric symptoms were not correlated.

Our results confirm the presence of emotion recognition deficit in PD and add new evidence to the debate concerning the pattern of impairment across different emotions (Argaud *et al.*, 2018). In line with the previous literature, by comparing a large sample of PD patients to healthy controls, we found preserved happiness recognition and a decline in all other emotions in PD (Argaud *et al.*, 2018; Gray & Tickle-Degnen, 2010). On the other hand, about one third of patients (24 out of 70) scored below the cut-off on fear recognition suggesting a disproportional deficit in recognizing fear, at odds with studies showing larger deficit in anger recognition (Clark *et al.*, 2008; Lawrence *et al.*, 2002; Lawrence, Goerendt, & Brooks, 2007).

The Ekman global score correlated with the severity of PD motor symptoms, the general cognitive abilities and tests tapping attentional, executive and visuospatial functions. Similarly, fear recognition correlated with PD stage, MoCA score, executive and visuospatial tests and resulted to be the expression with the lowest accuracy in our sample, according to normative data (Dodich *et al.*, 2014). Thus, it is plausible that the

severity of PD and the general cognitive profile significantly affect the ability to recognize the most difficult emotion. The MoCA score correlated also with anger, sadness and disgust recognition. The comparison between the three groups (healthy controls, patients with impaired/borderline MoCA scores and patients with higher MoCA scores) confirmed that the cognitive status affects patients' performance at the Ekman test. Cognitive impaired patients showed the lowest score and differed from the control group in all expressions, but happiness. Nevertheless, also cognitive unimpaired patients (MoCA ES > 2) performed significantly worse than controls, in particular in fear recognition. The relationship between emotion recognition performances and neuropsychological evaluation has been reported in previous studies, in particular for visuospatial, attentional and executive tasks (Assogna *et al.*, 2010; Narme *et al.*, 2013), however, correlations were not significant in other studies (Kan *et al.*, 2002; Marneweck *et al.*, 2014). Our data support the hypothesis that an emotion recognition deficit, mainly affecting fear emotion, exists in PD even in the absence of cognitive impairments, but the magnitude of the deficit increases with the progression of the disease and the emergence of cognitive symptoms (Argaud *et al.*, 2018). Notably, correlations with motor symptoms were not significant in different previous studies (Baggio *et al.*, 2012; Buxton, MacDonald, & Tippet, 2013; Clark *et al.*, 2008) and meta-analyses (Coundouris *et al.*, 2019; Gray & Tickle-Degnen, 2010), whereas our results, on a large sample of patients and applying bootstrap correlation methods, suggest a relationship between the Ekman global score and motor disability measured by the Hoehn and Yahr scale. On the other hand, a weak correlation emerged with the depression scale, whereas correlations were absent with scales on anxiety and impulsive-compulsive disorders, in line with the literature showing that emotion recognition deficits in PD are not related to mood disorders (Argaud *et al.*, 2018; Gray & Tickle-Degnen, 2010). Only the IRI subscale on empathic concern correlated with the Ekman global score and sub-score for sadness recognition in patients. This scale measures compassion for others, thus its relationship with emotion recognition, in particular the expression of sadness, suggests that patients with difficulties in expression identification suffer also from reduced empathy. Moreover, previous studies reported that the empathic concern subscale of the IRI was related to activity in the anterior insula and anterior cingulate cortex in healthy participants viewing pain stimuli and with atrophy in temporal and frontal regions in patients with neurodegenerative diseases (Rankin *et al.*, 2006; Singer *et al.*, 2004). Unfortunately, detailed brain images of our patients were not available, but previous studies showed correlations between emotion recognition performance and grey matter volume in the cingulate and orbitofrontal cortices in PD (Baggio *et al.*, 2012; Ibarretxe-Bilbao *et al.*, 2009), thus a more speculative interpretation could be that patients with lower scores in emotion recognition were affected by greater neurodegeneration in cortical regions crucially related to empathy.

Together with emotion recognition, this study aimed at assessing expressions discrimination using a backward masking paradigm with different presentation durations. This paradigm allows assessing sensitivity for stimuli processed with full awareness, but also for stimuli automatically processed, i.e., presented below the threshold for conscious perception (Pessoa *et al.*, 2005). The rationale for using different types of task was to evaluate multiple stages of emotion processing, i.e., sensory and visuo-spatial processes involved in forced-choice tasks as the backward masking paradigm, or identification and labelling processes involved in recognition tasks (Haxby *et al.*, 2000). Crucially, we did not find significant differences between PD and control groups in the emotion discrimination tasks. Only the confidence rating for hit trials was significantly higher in healthy than PD participants, showing that patients were less confident in judging their

performance although this was not impaired as compared to controls. Two different meta-analyses reported deficit both in emotion discrimination and recognition in PD (Coundouris *et al.*, 2019; Gray & Tickle-Degnen, 2010). In particular, previous studies reported impaired configural processing (Narme *et al.*, 2011) and discrimination of distinctiveness for faces in patients with PD, suggesting the hypothesis of a “cascade of lower-to-higher order impairment” for facial expressions, namely a critical contribution of visual sensory processes to deficits in emotion recognition in PD (Marneweck *et al.*, 2014). In contrast, different studies suggested unimpaired early visual and automatic emotion evaluation in PD (Alonso-Recio *et al.*, 2014; Castner *et al.*, 2007; Wagenbreth *et al.*, 2016; Wieser *et al.*, 2006). According to models of distributed neural system for face and emotion perception, different stages of facial expression processing are supported by partially distinct, albeit interactive, cortical and subcortical structures. Posterior occipito-temporal regions process perceptual information and are connected with subcortical structures, as the amygdala and basal ganglia, and frontal regions to elaborate emotional and social features (Haxby *et al.*, 2000; Ishai, 2008). The hallmark of PD neurodegeneration is the loss of nigrostriatal dopaminergic neurons, but also the amygdala and the prefrontal cortex are critically affected (Dauer & Przedborski, 2003; Diederich *et al.*, 2016). Indeed, neuroimaging studies with PD patients found that performances on the Ekman face test correlated with grey matter volume in the orbitofrontal and dorsal cingulate cortices (Baggio *et al.*, 2012; Ibarretxe-Bilbao *et al.*, 2009). On the other hand, a study with electrophysiological recording during facial expression recognition reported no diminished early visual processing, related to activity in posterior occipito-temporal regions, in patients with PD (Wieser *et al.*, 2006). Our data support these last findings, showing that PD patients' sensitivity for fearful and happy expressions, briefly presented between 17 and 334 ms, was not lower than control participants.

Interestingly, ACC and FA rates were higher for happiness than fear discrimination, as the  $d'$  index measuring perceptual sensitivity. Previous studies used backward masking paradigms to assess thresholds for visual awareness of fearful faces and to investigate the neural correlates of conscious and unconscious fear perception (Pessoa, 2005; Szczepanowski & Pessoa, 2015). To the best of our knowledge, performance in detecting fearful or happy target faces was not directly compared before. Our results showed higher sensitivity for happy faces, revealing no advantage for fear expression in automatic emotion processing (Quinlan, 2013; Roesch *et al.*, 2010; Williams, Morris, McGlone, Abbott, & Mattingley, 2004). The advantage for happiness recognition in the Ekman test could be related to the fact that it is the only entirely positive emotion. Surprise is also considered as positive but to a less extent, whereas the other possibilities are four negative emotions (fear, anger, disgust, sadness). The present data suggest that happiness is easier than fear expression also in a discrimination task, both for PD patients and control participants, in line with previous studies reporting unimpaired happiness discrimination (Kan *et al.*, 2002; Wagenbreth *et al.*, 2016).

Some limitations of the present study should be taken into account. Neuropsychological and clinical evaluation was not carried out for the control group of healthy participants. Thus, interpretations of correlations between emotion recognition and cognitive and neuropsychiatric profile are specific for PD group, whereas conclusions cannot be related to healthy participants. This would be of particular interest for the IRI scale, whose correlation with emotion recognition ability is not clearly supported by previous studies (Borgomaneri, Gazzola, & Avenanti, 2015; Kelly & Metcalfe, 2011; Riggio, Tucker, & Coffaro, 1989). Furthermore, we used two-dimensional static facial expressions; further studies with dynamic expressions will be crucial to investigate whether the

motor components of facial expressions impact on PD impairment in emotion recognition and discrimination.

In conclusion, from a theoretical point of view, our results revealed that PD affects emotion recognition, but not early perceptual stages of emotion processing. This suggests that emotional dysfunctions in PD could be related to deficit in declarative processes of emotion categorization, with intact automatic emotion discrimination (Argaud *et al.*, 2018; Wieser *et al.*, 2006). From a clinical point of view our findings highlight the relevance of an evaluation of emotion recognition in early stage of PD. Since this ability could be impaired even in cognitively preserved patients, a specific assessment is crucial to identify a deficit with potential impact on patients' social interaction in daily life (Argaud *et al.*, 2018). Moreover, PD is highly variable in its clinical phenotype with patients presenting different combination of motor, non-motor and neuropsychiatric symptoms (Erro *et al.*, 2013). These subtypes have been related to different progression of the disease, thus it could be relevant to include the assessment of emotion processing to better define the cognitive profile of patients and plan therapeutic interventions oriented to increase patients' quality of life (Alonso-Recio, Martín-Plasencia, Ruiz, & Serrano, 2018; Fereshtehnejad *et al.*, 2015).

## Conflicts of interest

All authors declare no conflict of interest.

## Author contributions

Giulia Mattavelli, PhD (Conceptualization; Formal analysis; Methodology; Writing – original draft) Edoardo Barvas (Data curation; Investigation; Writing – review & editing) Chiara Longo (Data curation; Investigation; Writing – review & editing) Francesca Zappini (Investigation; Resources) Donatella Ottaviani (Investigation; Resources) Maria Chiara Malaguti (Resources) Maria Pellegrini (Resources) Costanza Papagno (Conceptualization; Methodology; Supervision; Writing – review & editing).

## Data availability statement

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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Received 3 October 2019; revised version received 3 October 2020

### Supporting Information

The following supporting information may be found in the online edition of the article:

**Table S1.** Scores at neuropsychological tests included in patients' assessment.

**Table S2.** Clinical and neuropsychological data of 25 patients assessed with the backward masking paradigm.